



ACC.14

TCT@ACC-i2 | innovation in intervention

A1856

JACC April 1, 2014

Volume 63, Issue 12



TCT@ACC-i2: The Interventional Learning Pathway

IMPACT OF DIABETES MELLITUS ON CORONARY PLAQUE PROGRESSION AND CHANGE IN PLAQUE COMPOSITION IN NON-CULPRIT LESION UNDER OPTIMAL MEDICAL TREATMENT

Poster Contributions

Hall C

Sunday, March 30, 2014, 3:45 p.m.-4:30 p.m.

Session Title: Complexities and Complications

Abstract Category: 38. TCT@ACC-i2: Complex Patients/Comorbidities

Presentation Number: 2108-294

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Background: Diabetic patients continue to have high cardiovascular events after PCI under optimal medical treatment (OMT). However, serial changes in volume and tissue characteristics of coronary atherosclerotic plaques in non-culprit lesions have not been fully investigated. The aim of this study was to investigate the relationship between DM control, coronary atherosclerotic plaque progression, and change in plaque composition in non-culprit lesions using IVUS and integrated backscatter IVUS (IB-IVUS).

Methods: We investigated 37 patients with DM (39 lesions; DM) and 39 patients without DM (41 lesions; non-DM). Volumetric IVUS analyses were performed at proximal non-culprit 5mm lesions in de novo target vessels post PCI and at 6-12 months follow-up. All patients were managed with optimal medical treatment during follow-up. We measured serial change in coronary atherosclerotic plaque burden and plaque composition using IVUS and IB-IVUS.

Results: Baseline patient characteristics showed that the ratio of unstable angina pectoris (UAP), hypertension and dyslipidemia in DM were significantly higher than those in non-DM. Although DM demonstrated a greater plaque volume at baseline (DM: 41 mm³ vs. non-DM 33 mm³, $p < 0.01$) and follow-up (DM: 43 mm³ vs. non-DM: 33 mm³, $p < 0.0005$), plaque volume change showed no significant difference between groups. DM were further divided into 2 groups based on follow-up glycated hemoglobin (A1c) levels of $\geq 7.0\%$ (19 patients with 20 lesions) and $< 7.0\%$ (18 patients with 19 lesions). Serial change of plaque volume was significantly larger in A1c $\geq 7.0\%$ than that in A1c $< 7.0\%$ (+ 4.3 mm³ in A1c $\geq 7.0\%$, - 0.3 mm³ in A1c $< 7.0\%$, $p < 0.05$). Interestingly, serial change in LDL-cholesterol level induced by OMT significantly correlated with serial change of lipid volume in non-culprit lesions in DM ($r = 0.32$, $p < 0.05$), whereas it did not in non-DM ($p = 0.14$).

Conclusions: In diabetic patients, coronary atherosclerotic plaque progression was induced by poor glycemic control and high LDL-cholesterol level despite the use of OMT. Intensive medical treatment should be required for controlling non-culprit coronary plaque progression.